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Flatfoot and the Extension Boot Heel

The Use of a Stock Vaccine in the Preventive Treatment of Colds: A Report of 125 Cases

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Metal Fume Fever: II. Resistance Acquired by Inhalation of Zinc Oxide on Two Successive Days

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FLATFOOT AND THE EXTENSION BOOT HEEL*

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Akron, Ohio

ANTEDATING by many years the adoption of the modern foot plate, Hugh Owen Thomas (1) in 1878 published without written comment the picture of an extension boot heel now obsolete. The design was massive, extending from the back of the shoe to the distal metatarsal heads and filling in the hollow of the shank with solid leather. It traversed the inner half of the heel, shank, and sole, and apparently was beveled in a manner calculated to throw the body weight to the outer half of the foot. The logical inference is that his appliance was designed for the relief of certain types of painful feet then but poorly understood. Thomas deserves proper credit for instigating the present appreciation of the possibilities of the extended heel; his original pattern, however, has been universally and justly discarded because of its appearance, weight, rigidity, and mechanical failure.

Despite a voluminous literature during the past decade relating to painful feet there appear only scant and vague references to extension heels and sole wedges. Morrill (2) condemns the sole wedge, and Roberts (3), in his excellent paper relating to the malicious tilting of the os calcis, applies the principle of the inner heel wedge in the construction of his plates.

Something of the value of an extended heel has permeated in a vague manner the consciousness of the manufacturers of so-called "orthopedic" shoes and survived the commercial exploitation of the foot plate. Many of the corrective shoes purveyed to the public boast a modestly extended heel which has sacrificed the corrective and curative features

* Received for publication Dec. 13, 1926.

of an extension heel of proper construction. It is the purpose here to show that the correct heel is not replaceable by a rigid shank and that, rightly applied, it does possess advantages not inherent in any of the commercial heels or modern arch supports.

Coherence necessitates that certain accepted facts relating to the etiology of planus of the long arch of the foot be reiterated. Trauma, juvenile maldevelopment, and joint disease excepted, the outstanding causes of acquired flatfoot are those factors which compress the ball of the foot during walking. The popular glove fitting shoe into which the foot is crammed in the resting state does not permit action of the intrinsic muscles and spread of the forefoot during weight bearing, and as a result the forefoot is unconsciously turned outward and the weight is borne on the heel. The normal gait calling for physiologic activity of the arch in which the heel strikes first, followed by an inward thrust and grasping-like action on the part of the toes (Fig. 1), is unconsciously abandoned for a duck-like step in which the heel is implanted and the toes are held upward and outward in order to save the pinched forefoot (Fig. 2). The outward deflection of the forefoot is further enhanced by the usual outward curve of the inner border of the sole. The inner line of the foot is elongated, the curve of the long arch is flattened, and any attempt at utilization of the spring mechanism is hampered by compression and pain. Once the forefoot becomes widely abducted, the line of weight bearing falls inside the midline of the foot, the long arch descends, the scaphoid becomes prominent, and a persistent pronation develops at the ankle. Spasticity of the peroneal tendons indicates a constant disturbance of muscle balance, other secondary changes appear, and the early symptoms of foot strain are replaced by the familiar objective evidences of flatfoot (Fig. 3).

To approach the treatment with no other aim than to prop up a prolapse of the arch is to invite partial failure. Mechanical support of the planus assures an indifferent degree of comfort at the expense of any muscular activity and does not correct the pronation at the ankle or the abnormal and ungainly gait. Corrective exercises to restore muscle balance are of supreme importance, but they are irksome to the patient and are frequently practised irregularly and for short periods only. It is not to be denied that a combination of properly fitted shoes, well-adjusted arch supports, and carefully supervised exercises has been responsible for countless cures. Nevertheless, there are serious objections to the splinting of a physiologic structure in which the return of normal activity is the desideratum.

The extension heel can and does furnish a measure of support to the prolapsed arch but this is by no means its chief virtue. The tilt of the heel should slightly overcorrect any existing pronation (Fig. 4). The extension should project forward to a point under the articulation between the scaphoid and the internal cuneiform, or, for the benefit of the cobbler, to a point midway between the heel and the first metatarsophalangeal joint (Fig. 4). The forward extension

should have an elevation approximately $\frac{1}{16}$ inch greater than the broad dorsal portion (Fig. 4). This excess elevation of the forward end of the heel is of the greatest importance, for it forces a subconscious adduction of the forefoot with each step. As the weight is thrown forward, the high and extended portion exerts an upward and outward pressure under the midtarsal articulations and the forefoot is accordingly given an inward thrust. With the forefoot held in abduction, the elevated extension impinges against the ground in a manner that gives a feeling of instability with each step. The toes instinctively seek to correct this lack of balance by a grasping-like action which is essentially a corrective exercise (Fig. 5). Herein lies the chief value of the extension heel. Every step forces a muscular activity which is ultimately curative in that it compels a return to the proper mechanics of locomotion. Granting that the assumptions regarding the etiology of pes planus are correct, there are evident advantages in an appliance which corrects the pronation, adducts the forefoot, rectifies the unnatural gait, and makes of each step a corrective exercise.

Rigid arch supports, while lacking the curative properties of the extension heel, do furnish rest to overstrained tissues and exert a molding influence which is extremely helpful in beginning treatment of the third degree lesions. In the rarer cases where a midtarsal arthritis complicates a planus, the rigid support is indispensable.

The adducting effect of the extension heel on the forefoot requires some supervision, for a considerable degree of mesial deviation is obtainable. The infantile or congenital type of flaccid planus is very susceptible to any corrective force; especially when it is complicated by rickets, some degree of caution is necessary in order to avoid carrying adduction of the forefoot to a degree which will induce an inward rotation of the tibia. This inherent danger is, of course, of distinct advantage where the planus is complicated by an actual outward rotation of the leg. Overcorrection has not occurred in the treatment of several hundred adult cases and probably is not possible after ossification is complete.

Observations have been carried on largely among industrial employees where it has been possible to prescribe the modified heel and carefully to observe the final results. In order better to control the construction of the heels they were applied at the direction of the employer. Considerable experimentation was possible in these controlled cases as to the proper length and height of the extension, the angle of the lateral bevel, and the material. As so much of the corrective action is dependent on the excess height of the forward portion, an unusual effort was made to find a less abrasive material than leather. Rubber heels of a proper design have been produced and have proved extremely satisfactory.

The patients were directed to secure a broad shank shoe of the Munson type made of firm calfskin. Particular caution was enjoined in regard to the width. The feet of those patients who were suffering with severe spasm and pain were primarily strapped with adhesive and the patients were advised

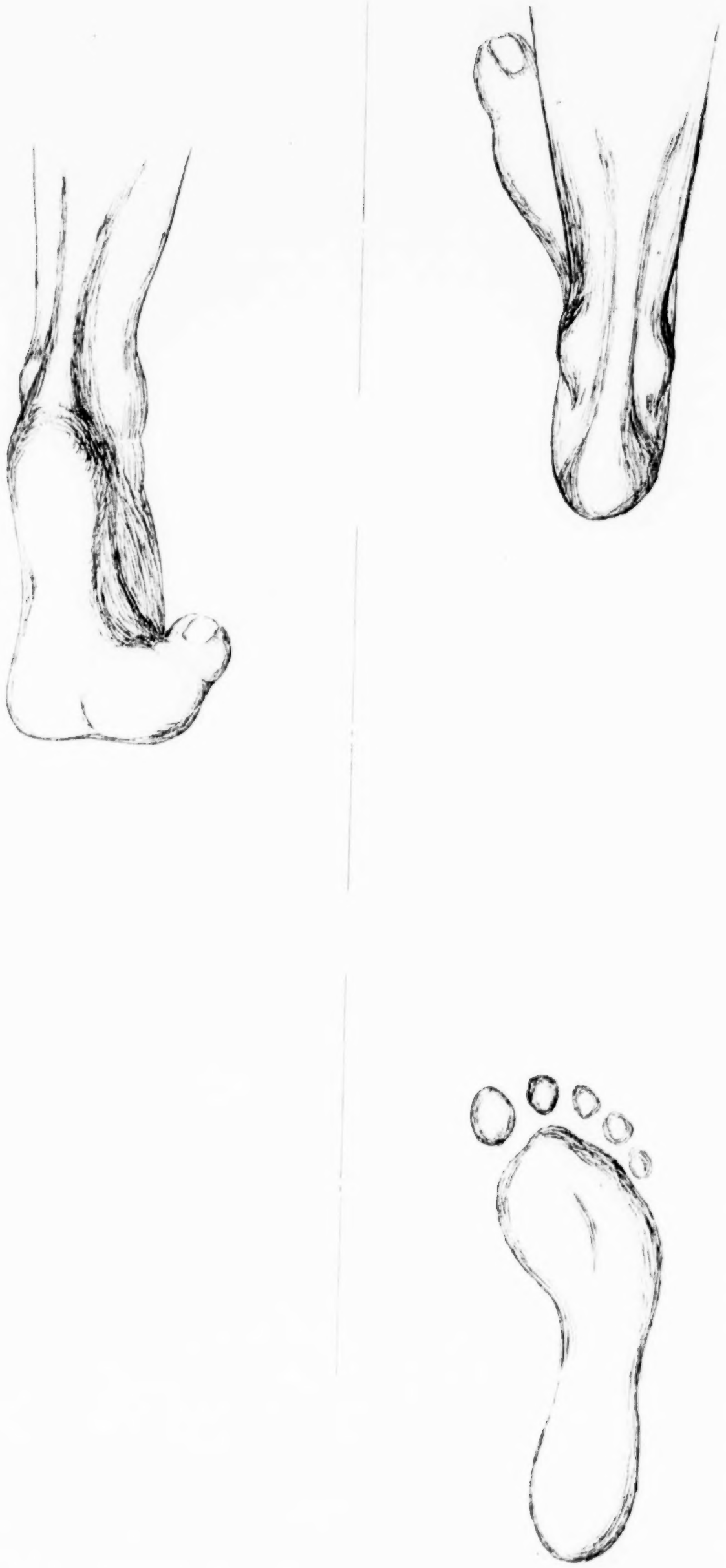

FIG. 1.—Normal activity with the grasping action of the forefoot enhancing the forward thrust.

FIG. 2.—The forefoot hampered by compression, grasping action lost, and the feet everted.

to wear the shoes for short periods only, until such time as they found the correction comfortable. Many were able to wear the heels imme-

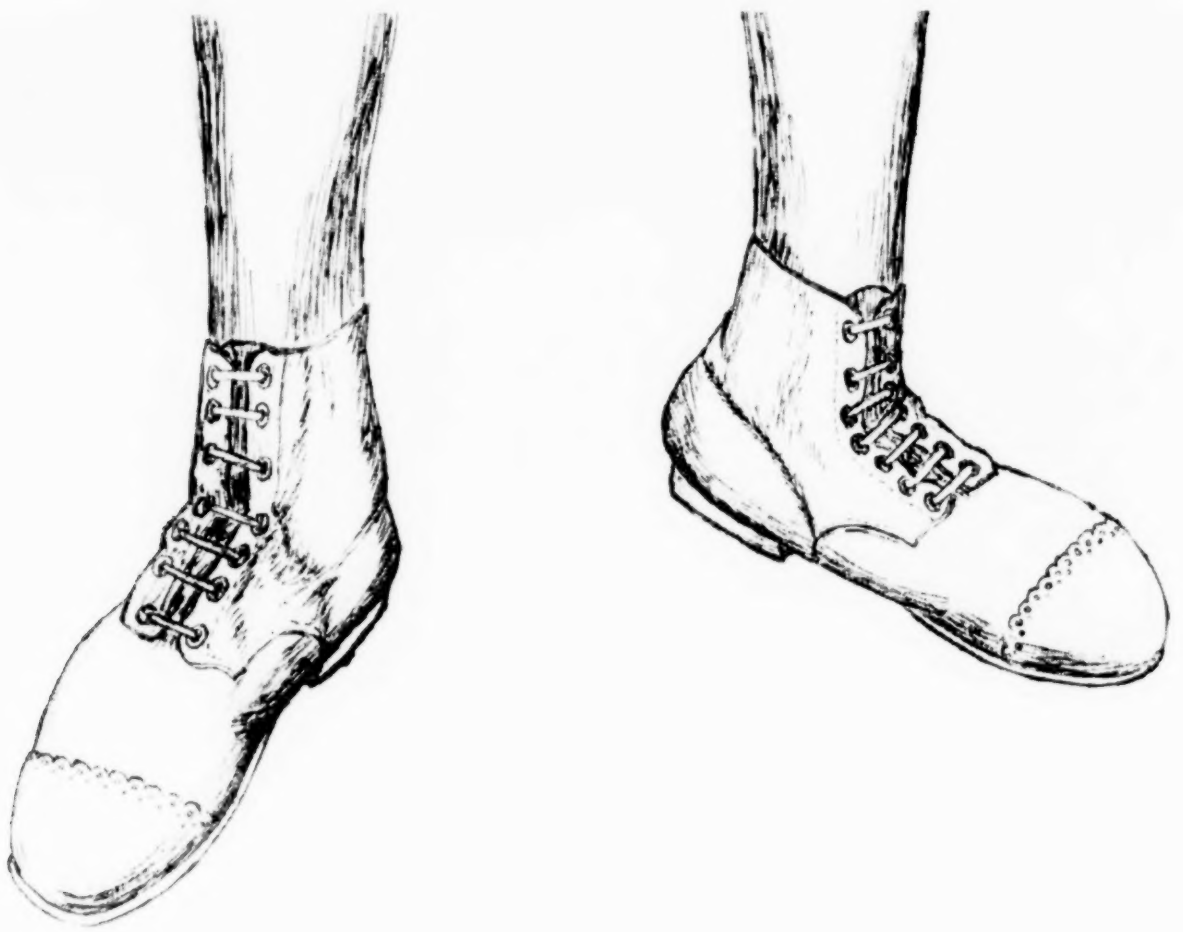

FIG. 3.—Abduction of the forefoot, prominence of the scaphoid, and eversion of the feet.

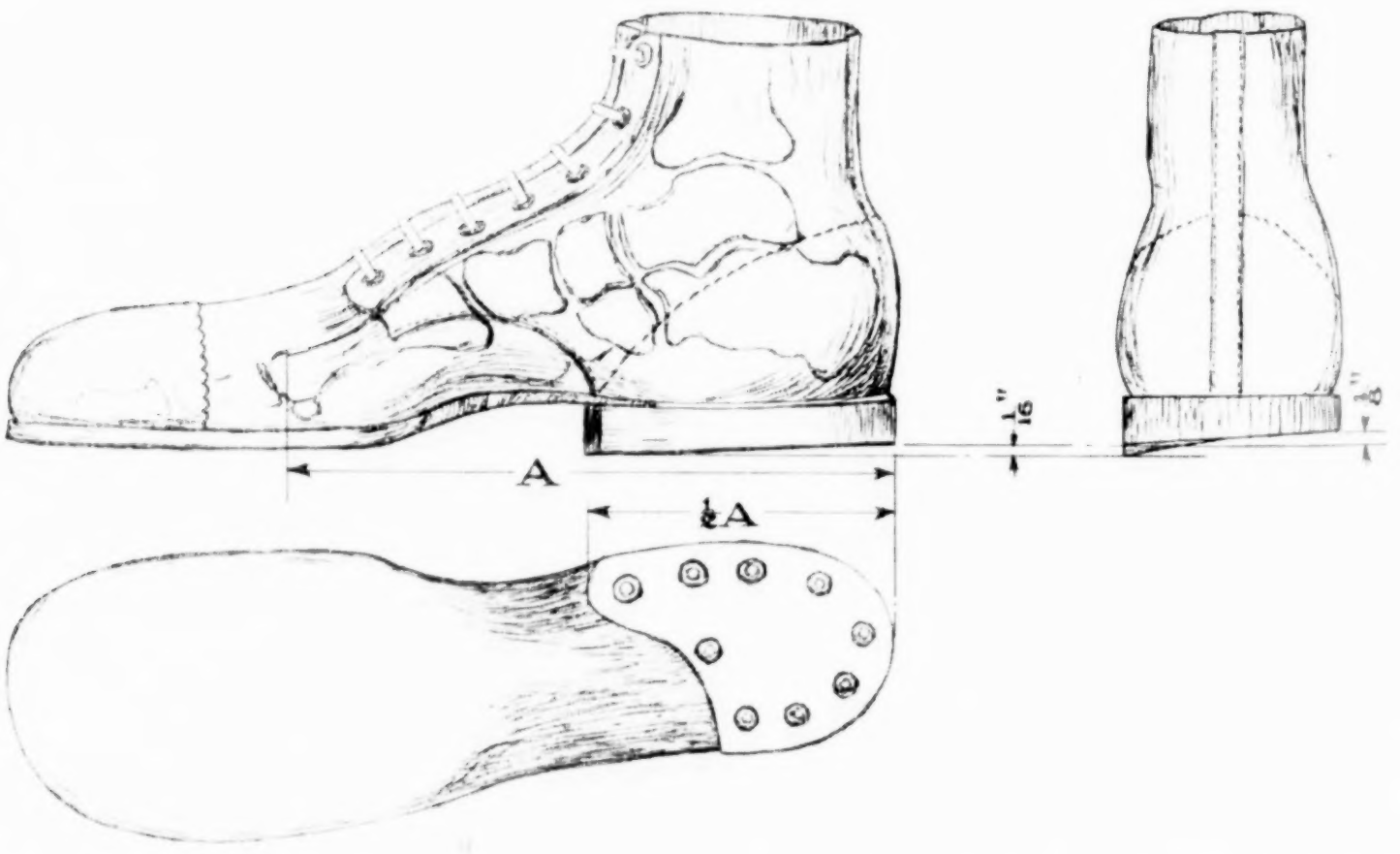

FIG. 4.—The mechanical details of the extension heel.

tely and constantly, while a limited number required, in order to adjust themselves, a period of from five to ten days during which the strapping was continued. Once the heels were continuously tolerated, no shoes other than those with the heels were permitted. The wearing of slippers or sneakers and walking in the bare feet were especially cautioned against.

The duration of treatment varied considerably with the individual patient and was influenced by the weight, the occupation, and the degree of planus. The majority of the patients had worn or were wearing arch supports of various types and, as might be expected, it was necessary to prolong the treatment of these patients. Also, those patients whose employment required long standing with little or no walking were noticeably slower in gaining correction. The degree of restoration of the arch proved to be of little value in determining the safe time for the discontinuance of the corrective heels. Permission for removal was granted when the gait evidenced a return of the propulsive activity of the long arch and the well-worn shoe disclosed a slight overadduction of the forefoot. An almost constant mesial deflection of the weight bearing line was noted to persist when the bare feet were observed, and on removal of the extension it was found advisable to prescribe a continuation of a beveled heel for an indefinite period.

Conclusions

The contraindications to the use of the extension heel may be limited to a small number of persistent third degree lesions in which correction is resisted because of bony deformity and pain and to all those cases complicated by a midtarsal arthritis.

Fig. 5.—The physiologic activity forced by the corrective heel.

The results observed and the almost unanimous relief admitted by the patients who have used extension heels have been quite in contrast to their early experiences with foot plates. The latter are of undoubted value when they are properly made and carefully supervised, but the time and expense involved are prohibitive where it is necessary to prescribe for large clinical groups. The extension heel is far more economical than plates are; it requires no careful adjustment and it is distinctly curative in that it forces a return of the normal muscular activity and support.

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J. I. H.
March, 1927

THE USE OF A STOCK VACCINE IN THE PREVENTIVE TREATMENT OF COLDS: A REPORT OF 125 CASES*

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THE QUESTION of lost time due to illness has ever been a thorn in the side of industry. Much of this illness is of the preventable type but it is always difficult to make much impression on a mass of people in a short time by educational methods only. Figures compiled by the state industrial boards seem to agree that the average loss of time per worker per year from preventable illnesses is about six days. In an effort to combat what is perhaps the most prevalent cause of this class of cases, the work here described was undertaken.

We recognize the fact that, theoretically, an autogenetic is to be preferred to a stock vaccine. In actual practice, however, this procedure is often impracticable for obvious reasons. We are confronted, then, with the proposition of doing the best we can with the tools readily available. We felt that a stock vaccine made by a reputable manufacturer and administered properly could do no harm. There was, on the other hand, a distinct possibility that it might be followed by good results, and so we felt no hesitation in proceeding with the inoculations.

* Received for publication Nov. 8, 1926.

In presenting the facts to the employees we made no promises. We stated our own beliefs and, on this basis, invited those who wished to try the inoculations to do so. We made a careful examination of the nose and throat in each instance before the vaccine was administered. This was done because we recognized the futility of our treatment in patients who were harboring grossly infected tonsils, or in patients in whom sinus, nasal, or postnasal involvement could be demonstrated. We excluded from our series those suffering from any form of allergy. This gave us a group of employees to work with who presumably were suffering throughout the winter from recurrent attacks of rhinitis due to their lowered resistance to the ever-prevalent bacteria of the respiratory group, *viz.*, influenza bacillus, Micrococcus catarrhalis, Friedländer's bacillus, and pneumococcus. Our vaccine was made up to include these offenders, together with several strains of Streptococci, and Staphylococcus aureus and albus.

The patients were selected and the inoculations were made in the early fall of 1925, so as to put the treatment to the rather severe test of a Philadelphia winter. Accurate records of each

patient were kept in the hospital files. The employees were instructed to report the recurrence of respiratory infection of mild or severe degree and this was promptly noted on the history card throughout the year.

At the end of this time, owing to the rather easy accessibility of the patients, we were able to collect and tabulate accurately the results of the completed inoculations. This we did by making a summary of the card histories; in addition to this we sent letters to the employees who had received the full series of inoculations, and their replies were checked with our case record cards. In this way we were able to estimate accurately the number who had reported to the hospital no recurrence of colds and to jog the memory of those who had neglected to report at all. We believe that we are in a better position to judge results than is the general practitioner or the hospital outpatient department, where disappointment is not uncommonly encountered owing to failure of the patients to report for follow-up examination.

From the 125 patients who were inoculated we received the following reports with regard to the recurrence of colds during a period of one year subsequent to the inoculations:

	Number of Cases	*Percentage*
No recurrence, apparent immunity	38	30.4
Some recurrence, but marked improvement in incidence	66	52.8
No improvement	21	16.8
Totals	125	100.0

Simey reports even better results and cites in his article an experiment similar to ours carried out in the boarding house of a large public school. This was even more encouraging than our result, since not one inoculated girl missed a single day at school on account of colds or influenza, while a considerable number of uninoculated controls were confined to bed with colds and influenza, and two contracted pneumonia. It is worthy of note in connection with our results that the absences, due to some form of upper respiratory disease, among the uninoculated employees during the winter of 1925, as reported by our Beneficial Society, were the highest they had been since the influenza epidemic of 1918.

The amount of vaccine given and the interval between the inoculations in our patients varied in each case according to the reaction of the individual. We usually started the series with an initial injection of 130 million of the mixed, killed bacterin and then administered at intervals of forty-eight hours 400, 600, 900, and 1,300 million. Reactions were not uncommon occurrences—in fact twenty, or 16 per cent., of the series experienced either local or general manifestation following one of the inoculations, usually the initial one. It is, however, interesting to note that of these twenty patients fifteen reported by letter that they enjoyed complete immunity from colds during the winter. The reactions, when they did occur, were, with few exceptions, local and mild and did not, as a rule, prevent the employee from following his usual daily routine.

Summary

1. Accurate follow-up of 125 patients inoculated in the fall of 1925 with a stock respiratory vaccine where examination demonstrated the absence of nose and throat infection shows thirty-eight, or 30.4 per cent. of the series, completely successful, with an additional sixty-six, or 52.8 per cent., demonstrating markedly increased resistance to upper respiratory involvement for a period of one year.

2. Reaction either local or general to the inoculations seems to be followed by a higher degree of immunity.

3. We consider the foregoing result sufficiently satisfactory to warrant continuation of the treatment this year.

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METAL FUME FEVER: I. CLINICAL OBSERVATIONS ON THE EFFECT OF THE EXPERIMENTAL INHALATION OF ZINC OXIDE BY TWO APPARENTLY NORMAL PERSONS*

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AND
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AT THE suggestion of Mr. Drinker and Mr. Thomson, who have been studying this subject for several years, two normal, healthy men were admitted for clinical observation to the Peter Bent Brigham Hospital immediately following the experimental inhalation of freshly generated zinc oxide.

CLINICAL HISTORIES OF SUBJECTS

The first subject was A, aged 32, Assistant Professor in the Harvard School of Public Health.

Family history showed no tuberculosis or other pulmonary infections.

Personal history was as follows: In general he has always been strong and healthy with the exception of a period of several months at the age of 14 years. At this time he developed a slight but persistent cough with a febrile reaction. Although sputum and roentgen ray examinations were negative, it was suspected that he might have pulmonary tuberculosis and he was sent to a camp in the mountains for two months. At the end of this time he completely regained his health and has never had a recurrence of his complaints. He has had several mild attacks of grip and four or five years ago had an attack of acute catarrhal jaundice.

This subject appeared for examination in July, 1926, as he had been inhaling zinc oxide frequently for experimental purposes during the past two years and he desired to know whether this had affected his physical condition. Following each experiment he had suffered only transient inconvenience, sometimes persisting for two or three days, and consisting mainly of a feeling of malaise. Subsequent to some of the experiments he had been troubled with pain and stiffness in the neck muscles which caused him some annoyance for several months.

Physical examination disclosed no abnormalities in any of the organs of the body. The patient appeared well nourished, robust, and in perfect health. The hemoglobin was 89 per cent. by the Dare method and a blood smear showed no abnormalities in the red blood cells, white blood cells, or platelets.

The second subject was C, Assistant in the Harvard School of Public Health, aged 39 years.

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Family history was irrelevant.

Personal history was as follows: In general he has enjoyed good health with the exception of several acute respiratory infections. Eight years ago he developed a sharp pain in the left side of the chest, related to breathing and associated with fever which kept him from his work for about three weeks. In 1917 he had a rather severe attack of influenza which incapacitated him for several weeks. Three years ago he developed a "bad cough" and was confined to bed for several days. For the past eight years he has been susceptible to respiratory infections, having one or two attacks each year, at which time he usually has a recurrence of "soreness" in the upper left part of the thorax, which occurred for the first time eight years ago. During the past two years this subject also has occasionally breathed air containing appreciable amounts of zinc oxide.

Method of Inhalation

For five days preceding this experiment, both subjects took care not to breathe abnormal amounts of dusts or fumes of any sort. This precaution was necessary in order to avoid the resistance said to be acquired by brass-founders and others if their exposure to metal fumes—particularly zinc oxide fume—occurs on successive days.

Zinc was brought to the boiling point in an electric muffle furnace and a gentle stream of oxygen was blown across the surface. The effluent zinc oxide[1] fumes passed into a 1,600 cubic foot (45 cubic meter) gas cabinet, producing in about ten minutes a cloud of sufficient density to be capable, during the next five hours, of producing fever.

At 7:25 A.M., one-half hour after completion of the fuming, both subjects entered the cabinet and remained seated during their exposure. The average concentration of zinc oxide, calculated as zinc, was 600 mg. per cubic meter while they remained in the cabinet. The subjects, who had both had the metal fume fever on several previous occasions, breathed the oxide for a length of time and in a manner which each knew would produce a typical reaction of only moderate severity. Subject A breathed for five minutes at the rate of 6 respirations a minute and for five and one-half minutes at 12 respirations a minute. As the result of previous experiments on the breathing of this fume, it is estimated that A inspired a total of about 78 liters of air, thereby inhaling 48 mg. of zinc, of which about 24 mg. were retained. C breathed for twelve minutes at a rate of 15 respirations a minute, inspiring a total of about 126 liters of air containing 74 mg. of zinc, of which about 37 mg. were retained.

Clinical Symptoms

The subjects were seen approximately one-half hour after the completion of the inhalations. At this time they stated that throughout the

[1] The zinc from which this oxide was made was furnished by the New Jersey Zinc Company, Palmerton, Pa. The analysis is as follows:

	%
Fe	0.0100
As	0.0002
Sb	0.0001
Al	0.0001
Sn	0.0010
Bi	0.0001
Cu	0.0002
S	0.0001
Pb	0.0620
Cd	0.0020
Zn (difference)	99.9242

exposure there was a tickling sensation in the nasal passages. At the end of five minutes they experienced a sweetish taste and developed a tendency to cough. The latter became marked by the end of the exposure and continued to a lesser extent for several hours after they left the cabinet. Coughing was induced by deep inspirations or forced expirations for a subsequent period of twenty-four hours. The sweetish taste remained for approximately one hour, at which time breakfast was eaten, and with this the abnormal taste disappeared. Immediately following the inhalation and continuing for several days, there was moderate substernal pain on coughing which was entirely similar to that of patients who are suffering from a tracheitis. About six hours after the inhalation C complained of nausea and anorexia. He ate a light lunch but no supper. A ate a lunch of average size four and one-half hours after the beginning of the experiment. Both subjects drank large amounts of water and orange juice.

For the first six hours following the inhalation both subjects complained of rather mild symptoms typical of the onset of an acute respiratory infection. These consisted chiefly of mild headache, malaise, backache, vague pains and sensation of "tenseness" about the eyes. At the end of six hours, both began to complain of chilly sensations and the body temperature became slightly elevated (99°F.). The chill never became an outspoken rigor in either case, but continued as a disagreeable feeling of coldness for several hours. The temperature curve of one subject, C, shows that the maximum febrile reaction (102.8°F.) was reached in four hours after the onset of the chill, and the body temperature came to normal between nine and twelve hours later. In the other subject, the body temperature reached its greatest height seven hours after the onset of the chill and reached normal in a period somewhere between six and nine hours later.

Examination of the chest of C showed a fairly large number of persistent fine, moist râles at the left base which were present one-half hour after the inhalation and remained unchanged for a period of three days. Eight days later a few transient râles of the same character remained in this same location. Examination of the chest of A showed no variation from normal.

Twenty-four hours after the inhalation, the patients' body temperatures were normal, but they both complained of headache, vague muscular pains, stiffness in the joints, and a general feeling of weakness and lack of energy. They also complained of a cough with substernal irritation on deep inspiration or forced expiration and raised a small amount of grayish mucoid sputum.

The pulse rates of both subjects throughout the period of observations showed no variation from normal except the usual and expected one which was associated with an increase in body temperature. The blood pressure, likewise, was not altered significantly (see Tables 1 and 2).

Leukocyte Count[2]

The white blood cell count is of considerable interest; the details are

[2] Earlier experiments by Dr. K. R. Drinker (1) led us to expect pronounced leukocytosis.

given in Tables 1 and 2 and are shown graphically in Figures 1 and 2. Between two and one-half and five and one-half hours after the subjects had inhaled zinc oxide, a moderate leukocytosis developed (averaging approxi-

TABLE 1.—SUBJECT C. CLINICAL DATA FOLLOWING INHALATION OF ZINC OXIDE

TIME	DATE	NUMBER OF HOURS AFTER INHALATION OF ZINC OXIDE	TEMPERATURE	PULSE RATE	LEUKOCYTE COUNT	VITAL CAPACITY	BLOOD PRESSURE	DIFFERENTIAL LEUKOCYTE COUNT				
								Polymorphonuclears	Lymphocytes	Large Mononuclears	Eosinophils	Basophils
			°F.	*per min.*	*per c. mm.*		*mm. Hg*	%	%	%	%	%
6:00 p.m.	Oct. 26	....	98.5	92	7,600	3,800	122/82	66	25	5	2	2
7:25 a.m. to 7:37 a.m., Oct. 27—inhalation of zinc oxide.												
8:00	Oct. 27	0.5	98.6	92	7,750	3,800	120/78	70	17	9	3	1
9:00	"	1.5	.....	...	.....	.....	.....	..	..	..	..	..
10:00	"	2.5	98.0	84	9,000	2,900	114/78	71	17	10	2	0
11:00	"	3.5	98.0	78	.....	.....	.....	..	..	..	..	..
12:00 noon	"	4.5	.....	...	.....	3,000	114/72	..	..	..	..	..
1:00 p.m.	"	5.5	98.2	80	11,400	.....	.....	80	16	2	0	0
2:00	"	6.5	99.0	100	11,250	.....	.....	85	11	2	1	0
3:00	"	7.5	100.0	100	10,000	1,800	120/80	..	..	..	..	..
4:00	"	8.5	101.5	96	.....	.....	.....	..	..	..	..	..
5:00	"	9.5	102.6	110	16,400	.....	.....	89	9	1	1	0
6:00	"	10.5	102.8	120	.....	.....	.....	..	..	..	..	..
7:00	"	11.5	102.1	108	13,000	1,800	.....	90	9	1	0	0
8:00	"	12.5	.....	...	.....	....	.....	..	..	..	..	..
9:00	"	13.5	100.4	90	10,000	.....	116/70	..	..	..	..	..
10:00	"	14.5	.....	...	.....	.....	.....	..	..	..	..	..
11:00	"	15.5	.....	...	.....	.....	.....	..	..	..	..	..
12:00 midnight	"	16.5	99.2	100	.....	.....	.....	..	..	..	..	..
1:00 a.m.	Oct. 28	17.5	.....	...	.....	.....	.....	..	..	..	..	..
2:00	"	18.5	.....	..	.....	.....	.....	..	..	..	..	..
3:00	"	19.5	99.1	80	.....	.....	.....	..	..	..	..	..
4:00	"	20.5	.....	...	.....	.....	.....	..	..	..	..	..
5:00	"	21.5	.....	...	.....	.....	.....	..	..	..	..	..
6:00	"	22.5	98.5	86	.....	.....	.....	..	..	..	..	..
7:00	"	23.5	.....	...	.....	.....	.....	..	..	..	..	..
8:00	"	24.5	98.6	96	16,850	3,400	110/66	80	16	3	1	0
12:00 noon	"	28.5	.....	...	14,500	3,200	.....	89	7	1	2	1
4:30 p.m.	"	33.0	.....	...	16,700	3,150	.....	74	20	4	2	0
9:00	"	37.5	97.6	...	9,750	3,400	.....	53	34	10	3	0
8:30 a.m.	Oct. 29	49.0	.....	...	6,800	3,800	.....	78	16	3	2	1
12:00 noon	"	52.5	.....	...	9,200	.....	.....	71	20	5	4	0

mately 12,000 cells per cubic millimeter). The maximum, averaging 16,600 cells per cubic millimeter, was reached in nine and one-half to eleven and one-half hours after the beginning of the experiment, at approximately

TABLE 2.—SUBJECT A. CLINICAL DATA FOLLOWING INHALATION OF ZINC OXIDE

TIME	DATE	NUMBER OF HOURS AFTER INHALATION OF ZINC OXIDE	TEMPERATURE	PULSE RATE	LEUKOCYTE COUNT	VITAL CAPACITY	BLOOD PRESSURE	DIFFERENTIAL LEUKOCYTE COUNT				
								Polymorphonuclears	Lymphocytes	Large Mononuclears	Eosinophils	Basophils
			°F.	*per min.*	*per c. mm.*		*mm. Hg*	%	%	%	%	%
6:00 p.m.	Oct. 26	....	98.5	84	6,000	5,900	108/76	70	24	2	2	2
7:25 a.m. to 7:35½ a.m., Oct. 27—inhalation of zinc oxide.												
8:00	Oct. 27	0.5	98.5	84	5,350	5,900	124/80	74	17	3	6	0
9:00	"	1.5	.....	..	.....	.....	.....	..	..	..	..	..
10:00	"	2.5	98.5	72	8,600	5,500	120/78	84	15	1	0	0
11:00	"	3.5	98.8	79	.....	.....	.....	..	..	..	..	..
12:00 noon	"	4.5	98.6	82	.....	5,300	118/70	..	..	..	..	..
1:00 p.m.	"	5.5	99.1	84	12,300	.....	.....	91	7	2	0	0
2:00	"	6.5	99.1	85	15,700	.....	.....	..	..	..	..	..
3:00	"	7.5	98.9	84	12,000	5,200	114/60	84	14	2	0	0
4:00	"	8.5	99.2	80	.....	.....	.....	..	..	..	..	..
5:00	"	9.5	99.6	80	12,200	.....	.....	88	10	2	0	0
6:00	"	10.5	100.0	86	.....	.....	.....	..	..	..	..	..
7:00	"	11.5	101.6	92	18,150	4,800	.....	93	6	1	0	0
8:00	"	12.5	.....	..	.....	.....	.....	..	..	..	..	..
9:00	"	13.5	101.8	98	12,000	.....	96/50	..	..	..	..	..
10:00	"	14.5	.....	..	.....	.....	.....	..	..	..	..	..
11:00	"	15.5	.....	..	.....	.....	.....	..	..	..	..	..
12:00 midnight	"	16.5	99.3	84	.....	.....	.....	..	..	..	..	..
1:00 a.m.	Oct. 28	17.5	.....	..	.....	.....	.....	..	..	..	..	..
2:00	"	18.5	.....	..	.....	.....	.....	..	..	..	..	..
3:00	"	19.5	100.0	82	.....	.....	.....	...	..	..	..	..
4:00	"	20.5	.....	..	.....	.....	.....	..	..	..	..	..
5:00	"	21.5	.....	..	.....	.....	.....	..	..	..	..	..
6:00	"	22.5	98.3	75	.....	.....	.....	..	..	..	..	..
7:00	"	23.5	.....	..	.....	.....	.....	..	..	..	..	..
8:00	"	24.5	98.3	98	16,650	5,300	112/60	91	9	0	0	0
12:00 noon	"	28.5	.....	..	16,400	5,200	.....	86	11	2	0	1
4:30 p.m.	"	33.0	.....	..	16,800	5,500	.....	78	18	4	0	0
9:00	"	37.5	97.9	..	10,300	5,800	.....	71	27	1	1	0
8:30 a.m.	Oct. 29	49.0	.....	..	8,800	5,900	.....	80	15	3	2	0
12:00 noon	"	52.5	.....	..	8,400	.....	.....	71	29	0	0	0

the same time as the febrile reaction. The leukocytosis persisted for an appreciable length of time after the body temperature reached normal, as the white blood cell count of both subjects was found to be over 16,000 per cubic millimeter about thirty hours after the inhalation had been taken, when the body temperature had been normal for almost eleven hours. The rise in the leukocyte count was due chiefly to the increase in the polymorphonuclear leukocytes as shown by the differential white blood cell counts in Tables 1 and 2.

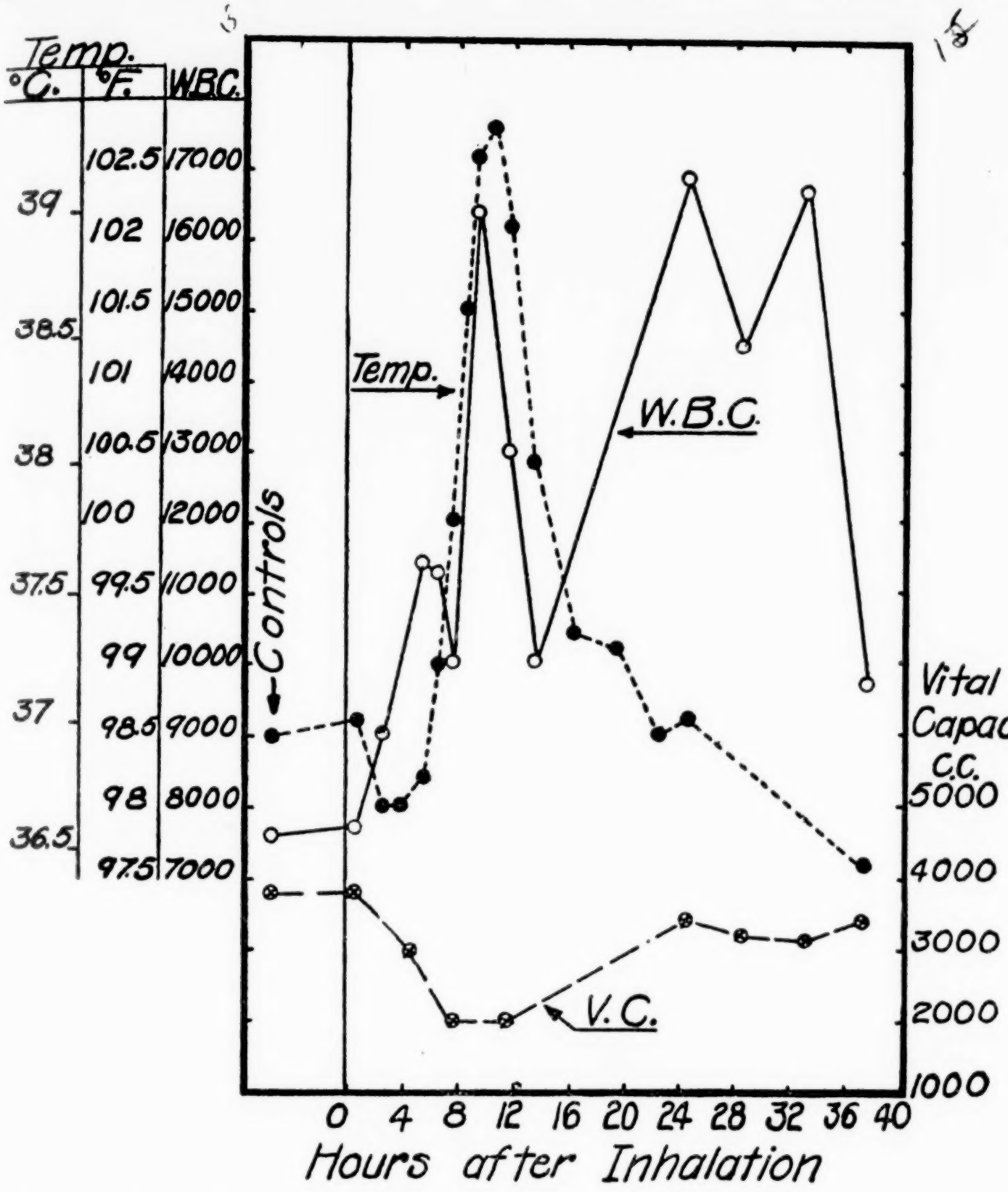

FIG. 1.—Subject C. Graphic representation of leukocyte counts, body temperature, and vital capacity from the data of Table 1.

It is interesting to note in both Figures 1 and 2 that there were apparently three phases to the leuko-

cytosis—which may be more than a coincidence. As shown by the curves of the white blood cell count, the initial leukocytosis was followed by a slight drop. A few hours later, a second rise in the white blood cell count occurred which was apparently in association with the maximum febrile reaction. There was then an immediate drop, but, before the normal level was reached, a third rise of considerable extent occurred which persisted for ten to twelve hours. This suggests the possibility that more than one factor, acting at different times, may be responsible for the leukocytosis. Of greatest importance is the third rise in the white blood cell count, which is present for rather a surprising length of time after the patient's temperature has returned to normal. It is possible

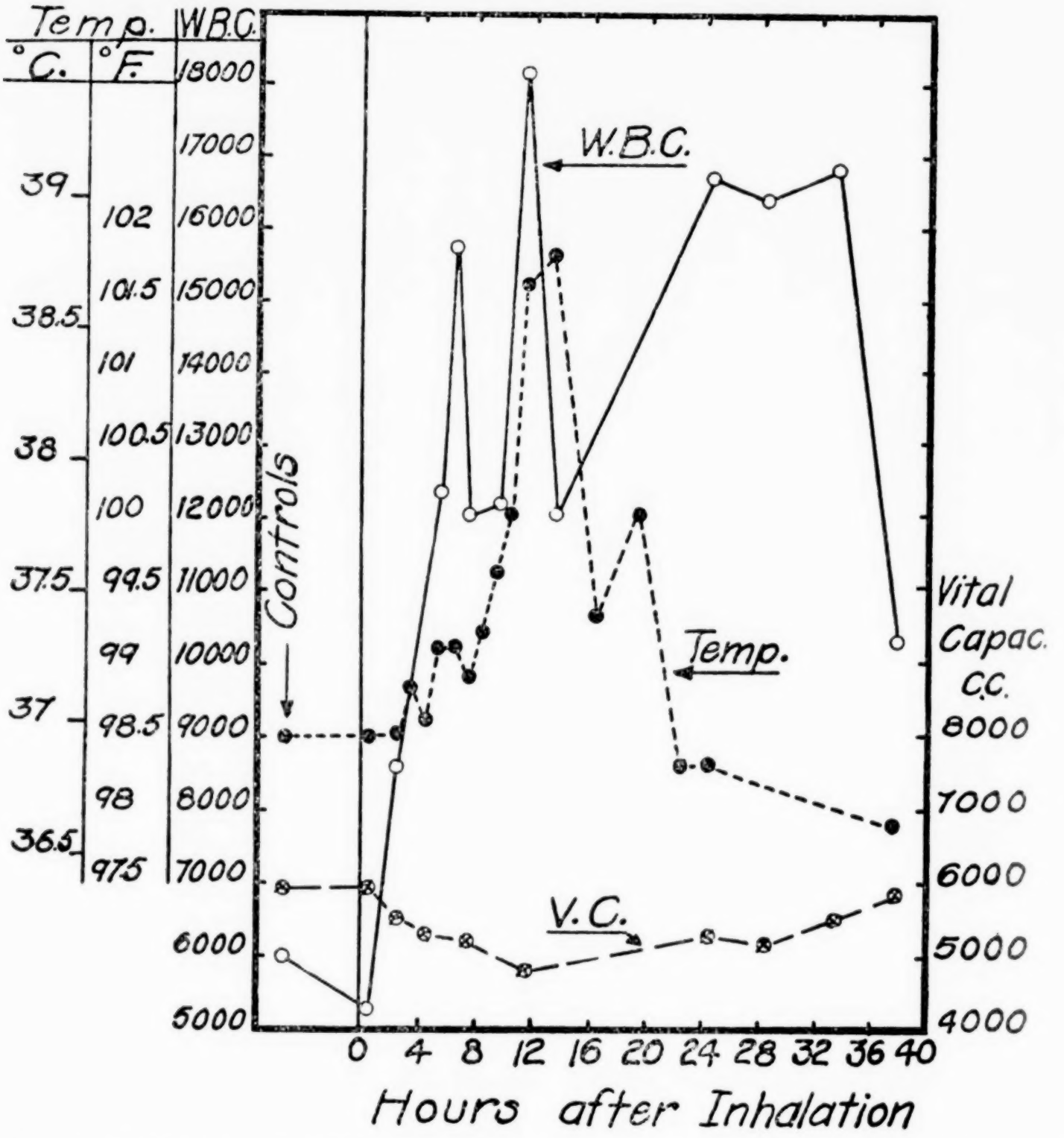

FIG. 2.—Subject A. Graphic representation of leukocyte counts, body temperature, and vital capacity from the data of Table 2.

that this leukocytosis may be associated with the reported resistance of workers who are exposed to fumes like zinc oxide on successive days. As far as we know, the persistence of leukocytosis for so long a time has hitherto not been observed, although it might have been suspected by Guelmann (2), who records blood counts of a typical fever case on the first and third days and thus missed the main reaction which, in our experiments, occurred on the second day. Also Arnstein (3) showed that a leukocytosis of some 17,000 followed about nine hours after exposure to zinc oxide fumes but he failed to record counts on the day following the exposures. In their study of twenty-four zinc workers, Batchelor, Fehnel, Thomson, and Drinker (4) found leukocyte counts ranging from 7,400 to 14,400. Of the twenty-four men examined, fourteen had counts over 10,000. All these men were exposed in their daily work to zinc dusts of varying concentrations. The persistent leukocytosis demonstrated in our experiment is of sufficient interest to warrant a special study on the relation between the resistance to reactions from metal fumes and the level of the white blood cell count.[3]

Vital Capacity of the Lungs

The vital capacity showed distinct changes which developed soon after the inhalation. Within two and one-half hours, the vital capacity in C had decreased approximately 24 per cent. and in A, 7 per cent. The maximum decrease was 53 per cent., which was observed in subject C and occurred in seven and one-half hours after the inhalation; after thirty-three hours, the vital capacity was still 17 per cent. below normal. In subject A, the changes were similar but less striking. He likewise showed an early decrease, but there was a maximum diminution of only 18 per cent., which occurred at eleven and one-half hours after the inhalation. At the end of thirty-three hours, there was still a decrease of 7 per cent. After forty-nine hours, both subjects had returned to normal.

The degree and course of these changes in vital capacity are consistent with what one might expect in transient irritations of the upper respiratory passages.

Roentgen Ray Examination of the Lungs[4]

Stereoscopic roentgen ray films of the chest taken on C about four months before the experiment showed no evidence of pulmonary tuberculosis. There was a slight clouding of the right costophrenic angle, probably due to an old pleurisy. Lung markings as a whole were more prominent than usual, possibly due to a bronchitis. Similar roentgen ray studies were made on this subject during the experiment, three and one-half hours after the inhalation of zinc, and again eight days later. No essential changes were observed from the appearance of the lungs on June 11, 1926, almost four and one-half months previously. Subject A had roentgen ray studies of the lungs on July 13, 1926, which showed the thorax, diaphragms, pleura, and heart to be normal. The

[3] See p. 98 of this issue.

[4] The roentgen ray studies were made by Dr. Merrill C. Sosman, Roentgenologist-in-Chief to the Peter Bent Brigham Hospital, Boston, to whom we are greatly indebted for his interest and assistance.

lungs were clear. The hilus shadows were somewhat more prominent than usual but their size and density were practically the same as in the roentgen ray studies made in December, 1920. Further observations on this subject three and one-half hours after the inhalation of zinc and eight days after the experiment showed no further changes. Roentgen ray studies, therefore, gave no positive evidence of value in explaining the nature of the changes induced in the lungs and bronchi following the inhalation of zinc.[5]

Observations on the Urine

All urine voided during the experiment and for two days following was collected and each separate voiding was studied carefully with reference to the color, reaction, specific gravity, presence or absence of albumin and sugar, and abnormalities in the sediment. No change from the normal was observed in any of the specimens. This observation under controlled laboratory conditions confirms the more extensive observations of Batchelor, Fehnel, Thomson, and Drinker (4) on zinc workers and of Drinker, Thompson, and Marsh (5) on animals. There is no reliable evidence that inhalation of zinc dusts or fumes in industrial processes cause kidney damage.

Clinical Interpretation of Data

Various symptoms which the subjects presented are easily explained. The irritation of the nasal passages and the substernal pain are undoubtedly due to the action of zinc oxide directly on the mucosa of the nose and trachea, as might be expected from the inhalation of any dry insoluble particulate substance. The vague aches and pains, headache, backache, and feeling of malaise are the classical prodromal symptoms of an acute febrile reaction. It is more difficult to understand why the vital capacity should be reduced to such an extent, but at least three factors may be of importance: (1) the irritation of the trachea (and probably the bronchi), which hampers deep breathing; (2) probably some bronchial spasm; and (3) a possible mild irritant action which extends to the alveoli of the lung and results in a slight exudative process with outpouring of a small amount of serum, fibrin, and red and white cells. This is substantiated somewhat by the occurrence of râles at the base of the left lung in subject C, although they were unilateral; moreover they were not present in the other subject. The fact that no changes were observed in the roentgenograms indicates that such a process, if it does occur, is not extensive.

The chill and subsequent febrile reaction is similar in many respects to the phenomena observed following the intravenous injection of typhoid vaccine, foreign sera, or blood for transfusion purposes. A similar reaction is likewise observed in association with cutaneous burns, whether they are due to the action of the sun or to other forms of heat. Lehmann (6) suggested in 1906 that the symptoms

[5] It should be recalled, however, that both of these subjects had repeatedly inhaled zinc oxide for experimental purposes and previous alterations in the lungs may have masked changes which occurred during the observed experiment. Against this is the fact that roentgenograms of the chest of subject A taken during the present experiment did not vary essentially from those taken six years ago, several years before he had inhaled zinc dusts.

following the inhalation of zinc oxide might be caused by the absorption of toxic protein products resulting from the action of zinc oxide on the lung epithelium—a reasonable assumption but one which is still unproved.

Summary

Two cases of metal fume fever following experimental inhalations of freshly generated zinc oxide are described. The subjects were observed clinically for fifty-two hours after their exposure. Typical febrile reactions occurred, and a marked leukocytosis followed the course of the fever and persisted for some twelve hours after the body temperatures had returned to normal. The vital capacities diminished as the temperatures increased; in one subject, the maximum diminution was 53 per cent., in the other 18 per cent. In both cases, this subnormality persisted for about fifteen hours after the fever had subsided. Changes in blood pressure were found not to be significant; roentgen ray examination of the lungs and analyses of the urine were negative.

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METAL FUME FEVER: II. RESISTANCE ACQUIRED BY INHALATION OF ZINC OXIDE ON TWO SUCCESSIVE DAYS*

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IN THE experiments described in the first paper of this series (1), it was found that a pronounced leukocytosis accompanied the metal fume fever produced by inhalation of zinc oxide and that this leukocytosis persisted for some twelve hours after the body temperature had returned to normal. This persistent leukocytosis suggested a possible explanation of the resistance which is said to be acquired by those who are frequently exposed to zinc oxide and certain other metal fumes.

The observations reported in the present paper were made on two normal, healthy, male subjects, A and C, whose medical histories were included in the first paper (1).

On the afternoon and evening (until 8:30) of April 23, 1926, subject C spent about five hours in a large brass foundry making determinations of the concentration of zinc oxide in the air. Subsequent analyses showed that the concentration he breathed averaged about 52 mg. of zinc per cubic meter of air. During the five hours of his exposure, he was performing the moderate physical exercise of carrying apparatus about, and the like. On returning to his hotel later in the evening (about 9:15), he was distinctly conscious that an attack of metal fume fever was coming on. He took a hot bath[1] and was in bed by 10:30. At 12:30 A.M. he awoke in a chilly, feverish condition and then slept intermittently until 7. Although no clinical thermometer was available to record his temperature, he undoubtedly had a typical case of metal fume fever. When he rose next morning he felt weak and tired but ate a good breakfast and returned to the factory a little before 9 A.M. to continue his work. On this second day he took his samples in a different part of the plant, near the electric furnaces where the fume concentration was generally high, the analyses showing that he was exposed to a concentration of about 330 mg. of zinc per cubic meter for some three hours. The work was rather hot and disagreeable, but he continued in spite of the fact that his

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[1] Guelmann's statement (2) that workmen believe a hot bath tends to abort the fever is not in accordance with our evidence (to be published later) that even protracted sweating fails of this effect. Before the onset of the fever, however, a hot bath is pleasant, just as it is in case of an anticipated febrile reaction of almost any sort.

fatigue augmented rather than abated during the progress of his work. That afternoon he anticipated either a second febrile reaction or a bad cold. He stopped work at 5 P.M., returned to his hotel, and ate a good supper. Before retiring he took a hot bath, then went to bed and slept soundly. The following morning he rose feeling well rested, ate a good breakfast, and returned to the foundry. On this, the third day, he suffered no inconvenience from the weakness and fatigue which had been so noticeable the day before.

The experience of subject C in the brass foundry and the experiments on subjects A and C previously described (1) persuaded us that more exact information in regard to acquired resistance could be secured if A were to undergo zinc oxide inhalations on two successive days under carefully controlled conditions.

Both exposures took place in our experimental gas cabinet, which was filled with zinc oxide fume generated by blowing oxygen across the surface of molten zinc. It was decided that A's exposure should be more severe on the second day than on the first. On both days, therefore, he breathed for eight minutes at 6 respirations a minute; but the average concentration on the first day was 430 mg. per cubic meter, the total inhaled approximating 23 mg. and the total retained about 12 mg.; whereas on the second day the concentration averaged 610 mg. per cubic meter, of which A inhaled 33 mg., retaining about 17 mg. Thus in his second exposure he breathed about 50 per cent. more oxide than in his first.

The most significant clinical manifestations of the fever in our previous experiments had proved to be the white blood cell count, the body temperature, and the vital capacity. Since reduction in vital capacity corresponded very nearly with the subjects' consciousness of their inability to inspire deeply and expire forcibly, such observations were omitted in this experiment. Observations on blood pressure, urine, and chest sounds, as well as roentgen ray examination of the lungs, had failed to show anything of significance, and so these also were omitted.

For five days previous to this experiment, A avoided breathing abnormal amounts of any dusts or fumes. Before his first exposure (at 10:09 A.M.) his temperature and white blood cell count were normal (see Fig. 1 and Table 1) and he felt in perfect health. After his exposure the usual symptoms developed, but were rather less severe than those of the former experiment (1) because his exposure was less severe. His temperature reached a maximum of 101.2°F. during the evening. The next morning he felt weak and debilitated and was conscious of a slight substernal pain on deep inspiration or forced expiration. These symptoms are a normal sequel to the fever.

On the second day he entered the cabinet again at 10:34 A.M. and, as stated above, breathed about 50 per cent. more zinc oxide than on the first day. The sweetish taste and tracheal irritation were present as usual. For the first two hours after leaving the cabinet he felt no indication of the oncoming fever. At the end of four and one-half hours, his temperature had risen to 99.5°F. and remained at

TABLE 1.—SUBJECT A. CLINICAL DATA FOLLOWING INHALATION OF ZINC OXIDE ON TWO SUCCESSIVE DAYS

TIME	DATE	NUMBER OF HOURS AFTER FIRST INHALATION OF ZINC OXIDE	TEMPERATURE	LEUKOCYTE COUNT	DIFFERENTIAL LEUKOCYTE COUNT					REMARKS
					Polymorphonuclears	Lymphocytes	Large Mononuclears	Eosinophils	Basophils	
			°F.	per c. mm.	%	%	%	%	%	
9:10 a.m.	Nov. 22	....	98.5	5,450	70	25	4	0	1	Felt in perfect health.
10:09 to 10:17, Nov. 22—first inhalation of zinc oxide.										
10:20	Nov. 22	0.3	.....	9,150	72	21	5	2	0	Sweetish taste.
10:30	"	0.5	99.1	.....	....	....	....	....	....	.....
11:35	"	1.6	.....	8,350	69	24	5	1	1	.....
11:55	"	1.9	98.8	.....	....	....	....	....	....	.....
1:00 p.m.	"	3.0	.....	8,400	73	22	3	2	0	Ate normal lunch.
2:00	"	4.0	98.7	.....	....	....	....	....	....	Conscious of oncoming fever.
2:10	"	4.2	.....	8,950	72	23	3	2	0	.....
3:45	"	5.8	.....	9,850	68	25	4	2	1	.....
4:15	"	6.3	98.6	.....	....	....	....	....	....	Went home.
5:10	"	7.2	.....	13,000	71	24	4	1	0	.....
5:45	"	7.8	98.8	.....	....	....	....	....	....	Ate normal dinner.
7:10	"	9.2	.....	14,850	81	16	2	1	0	.....
8:30	"	10.5	99.2	12,350	79	18	2	1	0	Chilly. Went to bed.
9:00	"	11.0	98.8	.....	....	....	....	....	....	.....
10:00	"	12.0	99.8	.....	....	....	....	....	....	Feverish.
10:10	"	12.2	.....	12,250	83	14	2	1	0	.....
10:40	"	12.7	100.5	.....	....	....	....	....	....	.....
11:15	"	13.3	101.2	.....	....	....	....	....	....	.....

1:00 a.m.	Nov. 23	15.0	101.0	12,400	86	10	3	1	0	.....
4:00	"	18.0	98.7	15,500	85	8	6	1	0	Weak and somewhat debilitated.
8:15	"	22.3	.....	19,000	84	11	4	1	0	Ate normal breakfast.
9:00	"	23.0	98.5	.....	....	....	....	....	....	.....
10:30	"	24.5	.....	17,200	83	14	3	0	0	.....
10:34 to 10:42, Nov. 23—second inhalation of zinc oxide.										
10:50	Nov. 23	24.8	98.3	15,450	83	12	3	1	1	Sweetish taste.
11:45	"	25.8	.....	13,150	77	19	3	1	0	.....
11:55	"	25.9	98.8	.....	....	....	....	....	....	.....
1:00 p.m.	"	27.0	.....	11,200	78	18	3	1	0	.....
2:25	"	28.4	.....	17,000	79	18	2	0	1	.....
2:55	"	28.9	99.5	.....	....	....	....	....	....	Tired but not feverish.
3:21	"	29.4	99.4	.....	....	....	....	....	....	Slightly feverish.
3:30	"	29.5	.....	11,750	84	11	4	1	0	.....
4:05	"	30.1	99.5	13,200	79	15	4	1	1	Went home.
5:20	"	31.3	99.5	11,000	86	8	5	1	0	.....
6:00	"	32.0	.....	.....	....	....	....	....	....	Ate light supper.
6:35	"	32.6	99.5	.....	....	....	....	....	....	.....
8:10	"	34.2	99.3	.....	....	....	....	....	....	Played cards. Comfortable.
9:40	"	35.7	99.4	.....	....	....	....	....	....	Played cards. Slightly chilly.
10:10	"	36.2	.....	13,150	76	18	5	1	0	.....
10:35	"	36.6	99.5	.....	....	....	....	....	....	Went to bed and fell asleep immediately.
2:40 a.m.	Nov. 24	40.7	98.0	.....	....	....	....	....	....	Slightly fatigued but much refreshed.
6:15	"	44.3	98.0	.....	....	....	....	....	....	Felt about normal.
9:35	"	47.6	.....	10,550	74	21	3	1	1	Felt about normal.
12:30 p.m.	"	50.5	.....	11,950	80	14	5	1	0	Entirely recovered.
5:50	"	55.8	.....	10,150	67	27	4	1	1	.....
9:25 a.m.	Nov. 25	71.4	.....	4,600	69	25	4	2	0	.....

that point for somewhat more than eight hours. Until 4 P.M. the subject went about his customary work in the laboratory but was conscious of a slight malaise and a feeling of weariness. During the next six hours he remained at home, ate a light supper, and played cards until 10:30 P.M., when he went to bed with a temperature of 99.5°, fell asleep at once and slept soundly until 2:40 A.M. He was waked at that time to take his temperature, which proved to be 98°. He rose at 7 A.M. feeling much refreshed, with normal temperature and with none of the sensations of weakness that he had always experienced on the morning after an attack of metal fume fever.

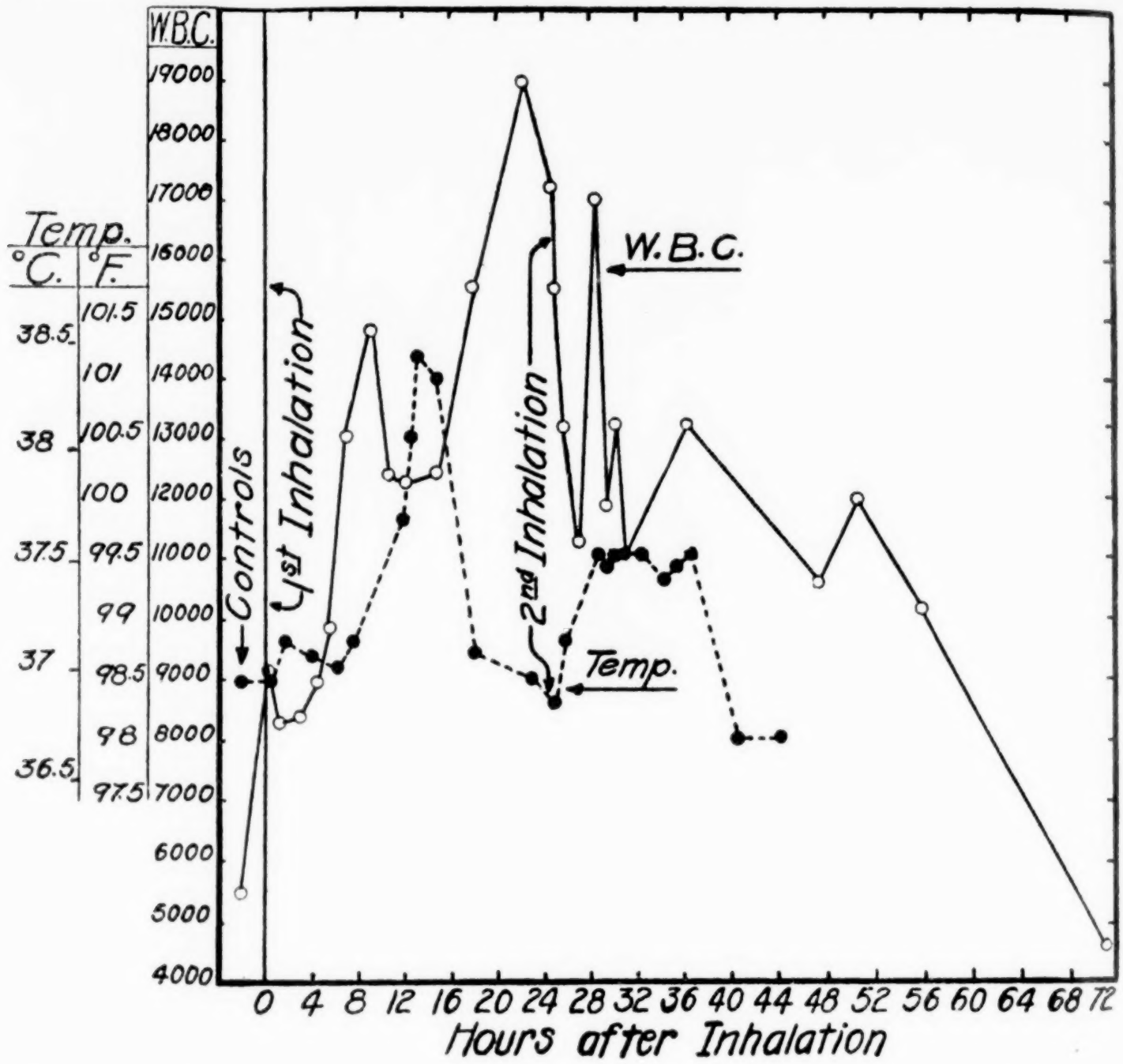

FIG. 1.—Subject A. Graphic representation of clinical data following inhalation of zinc oxide on two successive days, from Table 1.

The leukocyte count of A is given in Table 1 and is illustrated graphically in Figure 1. The leukocytosis produced by the first inhalation follows the same general course as that formerly reported (1). The second inhalation took place, by design, while the white blood cell count was still high (16,000) but was presumably returning to normal when the second inhalation intervened. About four

hours after the second inhalation, the count again rose abruptly to 17,000, fell to 11,750, and then remained at over 10,000 for the next twenty-seven hours, persisting for some fifteen hours after the slight fever (0.8° to 1°F.) had abated. From the differential counts, Table 1, it will be seen that the increase in the number of leukocytes following each inhalation is owing mainly to an increase in the number of polymorphonuclear cells. This is in agreement with the results of the experimental inhalations described in our previous paper (1).

Discussion

In the casting of brass, metals other than zinc are found in the fumes in amounts which vary approximately in proportion to the partial pressure of the metals composing the melt and to their respective heats of oxidation (3).

The fume breathed by C contained traces of cadmium, iron, and lead, and 0.6 per cent. of copper. We believe that the amounts of these impurities were too small to be of physiologic significance, although, if present in sufficient quantity, each of these elements or its oxide in itself produces characteristic reactions.

The fume breathed by A was perhaps not strictly "chemically pure," but it contained less than 0.08 per cent. of impurities.[2] Workers in the plant where this particular metal is refined show none of the effects ordinarily associated with continued exposures to lead or cadmium (4). In the case of A, therefore, we feel secure in ignoring the effect of the impurities present in the oxide.

[2] Analysis is given in full in (1), footnote 1.

The two inhalations of A, each for eight minutes at 6 respirations a minute, were brief and so perhaps were not representative of the exposures commonly met with in industry. Moreover 6 respirations a minute is an abnormally slow rate of breathing. In many previous experiments, however, we have found that slow breathing (and hence deep breathing) is vastly more effective in producing the fever than a normal rate of 12 to 18 respirations a minute. Since breathing these fumes is never pleasant and it is desirable to make the exposure as brief as possible, we usually adopt this slow rate in our experiments.

The data (5) which we have available on A, as a result of eight attacks of the fever, show that his second inhalation ordinarily would have produced a fever of about 102.5°F. (39.2°C.)—a little more than 1° higher than the fever caused by his first inhalation. In the case of C, the complete data are lacking; but, as he had experienced the fever on several occasions before and was thoroughly familiar with its symptoms, it seems reasonable to assume that, without the desensitizing effect of his first exposure, his second exposure would have produced a severe reaction. Among brassfounders it is almost axiomatic that a good attack of fever on one day renders the victim resistant to an attack on the next. Indeed, it has often been pointed out in the literature that workers are more susceptible to the fever on Mondays and on week days following a holiday than on other working days. This is in accordance with the evidence offered in this paper.

Guelmann (2) has recorded a severe

case of fever in a brass caster. At the peak of the fever, the subject's temperature was 104°F. (40°C.), and two days later his white blood cell count was 11,400 compared with 7,650 (presumably his normal count) on the day of his exposure. In a case of such severity it is possible that the acquired resistance may continue into the third day, but we have no data to prove this point.

In experiments on animals, Drinker and her associates (6) have obtained evidence which indicates that an interval of four days is normally sufficient for the removal of zinc oxide inhaled into the lungs, even when the exposure is many times more severe than that which we consider safe for man. In experiments on men, therefore, we normally allow an interval of at least five days between exposures and our results show that no desensitizing effect remains at the end of that time.

Although we cannot say that A's leukocytosis on the day following his first inhalation was the sole cause of his resistance to the second exposure, the white blood cell counts appear to be the most "easily measured indicators of resistance" (7) to metal fumes.

It would seem that the whole question of the relation between the level of the white blood cell count and the elimination of inhaled insoluble dusts and fumes of all sorts could be studied with profit. We plan further studies on this question.

Summary

"Resistance acquired by inhalation of zinc oxide fume on successive days is reported in two typical cases."

In the first case, the subject was exposed to air containing a concentration of about 52 mg. of zinc per cubic meter of air and the next day was resistant to a three-hour exposure in air containing 330 mg. of zinc per cubic meter.

In the second case, another subject inhaled 23 mg. of zinc (as zinc oxide) and as a result developed a fever of 101.2°F. (38.4°C.). A second inhalation of 33 mg. of zinc under like conditions but in a heavier fume concentration caused a slight malaise and raised the body temperature to a maximum of 99.5°, where it remained for about eight hours. It is estimated from former experiments on this subject that, without acquired resistance, his temperature would have risen to about 102.5°F. (39.2°C.). A pronounced leukocytosis was caused by the first inhalation and, while the white blood cell count was still high (16,000), the second inhalation took place. The count then remained at over 10,000 for the next twenty-seven hours.

"On the day after their first inhalations of zinc oxide, both subjects experienced the weakness and debilitation that follow typical attacks. On the day after their second exposures, both were free of these symptoms and felt themselves to be in normal health."

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BOOK REVIEW

HANDBUCH DER SOZIALEN HYGIENE UND GESUNDHEITSFÜRSORGE. Edited by *A. Gottstein*, *A. Schlossmann*, and *L. Teleky*. Vol. 2, Gewerbehygiene und Gewerbekrankheiten. Paper. Pp. 816, with 56 illustrations. Berlin: Julius Springer, 1926.

With the rapid progress of the various branches of industry the significance of industrial hygiene and industrial disease is continuously increasing. While thorough knowledge of these branches of medicine requires specialization, certain familiarity with the field is essential for all the members of the profession. There is no text which treats the subject as precisely, as thoroughly, and from as many angles within 816 pages, as this work. It should serve as a source of information not only for those primarily interested in this field, but also for physicians in general practice. Individuals who are concerned with the technical aspect of industrial hygiene will find a classical account of the clinical side of the problem, and physicians in clinics will find the book of value from the point of view of the community. There are seventy-seven chapters written by thirty-five contributors, each of whom is an authority on his topic. With the characteristic thoroughness of the earlier German medical writing, each topic is introduced with a short historical account. Each problem starts with elementary definition, and the material is built up systematically in a clear and easily understandable way. Numerous diagrams and pictures illustrate the more complicated technical points and the rarer clinical manifestations of disease. There are references to all the important contributions in the field.

The work presents facts and problems related to human beings only. Experimental evidence is purposely omitted to a large extent. As Teleky points out, although experimentation is valuable in certain problems, it often rather confuses the issue. Susceptibility of animals to a certain poisoning as compared to that of man is difficult to determine.

The first part of the book (pages 1 to 203) is concerned with the general topics of industrial hygiene and of industrial diseases. Teleky calls attention to the difficulties connected with generalization and interpretation of either statistical or clinical findings. In studying industrial diseases it is important to remember the entire body rather than a part of it. Early diagnosis, as far as successful treatment is concerned, is just as important in industrial diseases as in any other diseases. This early recognition of industrial diseases depends on training. Symptoms of intoxication almost invariably precede signs. Certain intoxications manifest themselves by symptoms only. The symptomatology is therefore relatively more important in industrial diseases than in other branches of medicine. The symptoms and signs of the same industrial intoxication vary greatly. Because industrial poisoning is often connected with lawsuits for compensation, objective (laboratory) procedures to prove clinical diagnosis are of great importance. Examples illustrating this point are mentioned.

The duties of physicians in maintaining proper industrial hygiene are enumerated by Teleky. Specific descriptions of methods used in larger German industrial centers are given. A short summary of legal control of industries in several of the larger industrial countries of the world is given by Brezina. The influence of the English plan, adopted as early as 1802, on other European countries is evident. The first country that followed England in controlling child labor was Prussia.

The Taylor system is discussed in detail by Brezina. In the controversy as to whether Taylor was a capitalist or an apostle bringing blessing to the suffering labor class, the author of the chapter agrees with those who believe that Taylor was a bigoted capitalist who, however, was wise enough to recognize that the success of capital depends on the co-operation of the labor class.

Betke gives a good discussion on the influence of occupation on sexual life. Statistics quoted from larger communities of Germany show that such a relation exists. Sexual aberrations peculiar to certain trades are mentioned.

The first part of the book ends with a description of technical equipments for factories which are of hygienic importance.

The second part of the book (pages 203 to 600) is devoted to specific industrial pathology and industrial hygiene. Koelsch emphasizes the fact that the reliability of morbidity rate statistics is still suffering from the inadequate records of physicians attending workmen. According to the statistics quoted, the morbidity of female workers is higher than that of male workers of identical age. The difference is marked between the ages of 25 and 35. The reason for this increased difference is the double duties of the female workers as laborers and mothers. The high mortality rate among laborers inhaling inorganic dust particles in Holland is striking.

Joachimoglu, the pharmacologist, gives a very good general account of the fate of poisons in the human body, and discusses the significance of the

various channels of the body for entry and for elimination of poisons. The different mechanisms of habituation to poisons probably have important rôles in the development or in the absence of industrial intoxications in a given individual. Detailed description is given of the mechanism and of the manifestation of industrial diseases induced by various organic and inorganic agents. Among these chapters an excellent one is that by Teleky on lead poisoning. Within forty-four pages he discusses the fate of lead in the body, the usual manifestation of lead poisoning in different organs, and the different factors predisposing to lead intoxications. The clinical picture of patients suffering from poisoning by tetraethyl lead is given. It is emphasized that, aside from the acute intoxication, some patients manifest symptoms of slow development as a result of exposure to tetraethyl lead. The rôle of the induced arteriosclerosis in such patients is not yet clear. All symptoms and signs of lead poisoning, with the exception of the weakness of the extensor muscles, have a tendency to disappear when the condition becomes chronic. As to the therapy, the author accepts the suggestions of recent American investigators.

Sellner discusses the effect of exposure to electric currents. Late neurologic complications due to electric shocks are clearly shown both by the general discussion and by case reports. The author therefore does not think that the late neurologic complications can be explained as hysterical manifestations, an opinion defended by certain authorities. The mechanism of acute death after electric accidents is still obscure. Among the therapeutic measures applied after severe shock, the application of artificial respiration is the most important.

Among the infectious diseases, anthrax, glanders, epidemic stomatitis, and smallpox are discussed as occupational diseases. Among the parasitic infections, hookworm disease is treated in detail. The discrepancy between the general spread of the disease among the population, regardless of occupation, within a belt of 30 to 35 degrees on both sides of the equator, and the selective infection of tunnel workers and of miners in areas of higher latitude, can be explained by the discovery that the disease is spread through the fully developed larvae. For the development of the larvae a certain temperature, which is present above ground in the tropics and underground in higher latitudes, is essential.

In the chapter on the relation of trade to malignant tumors, statistics are quoted showing suggestive differences between different occupations. Koelsch believes that there is a definite relation between frequency of carcinomas of the skin and occupations associated with continuous exposure to light and to weather changes. A similar relation exists between skin cancer and occupations which give opportunity for exposure to "chemical rays." According to the present conception the traumatic factor in precipitating malignant tumors is not of such importance as it was considered to be in the past, though its significance is definite. Traumatic factors are rather important in the etiology of bone sarcomas. The frequency of primary carcinomas of the lungs

among the mining population of Schneeberg, Saxonia, must have relation to inhalation of particles of cobalt and of arsenic. There are 250 primary tumors of the bladder reported in the German literature among aniline workers.

Within 100 pages, the influence of occupation on tissues and on organs is discussed. Aside from the organic changes in the tissues the functional alteration of organs without evident anatomic changes is described. The chapter on the effect of occupation on the skin, by Oppenheim, is richly illustrated.

The third part of the book (pages 600 to 785) describes the hygiene of trades and occupations. A detailed account is given of the daily activities of laborers in different trades and of the actual and potential factors influencing their bodily activities during their work. Accident hazards connected with certain work are described, and methods for prevention and for treatment are mentioned. There are critical discussions as to whether certain measures are adequate or not, and in what direction improvement should be searched for. Schurmann quotes enlightening statistics with regard to the activities of coal miners. The advantages of the uniform scheme adopted in Germany since 1924 (Reichsknappschaftverein) for the general welfare of the miners, for uniform miners' insurance, and for the care of the sick and invalid are discussed.

The authors of the book should be congratulated. They have presented a valuable contribution to the medical profession.—*Soma Weiss.*